WHAT IS CLAIMED IS:

1. A method for identifying a therapeutic agent for use in treating a CAR-mediated disorder or condition, the method comprising:

identifying a candidate therapeutic agent by screening one or more compounds to determine whether said compounds can modulate a CAR-mediated intermolecular interaction;

administering the candidate therapeutic agent to a test mammal; and

determining whether the level of a cholesterol indicator is modulated in said test mammal.

2. The method of claim **1**, wherein said candidate therapeutic agent is 5β-pregnan-3,20-dione.

3. The method of claim **1**, wherein said CAR-mediated disorder or condition is selected from the group consisting of: hypercholesterolemia, lipid disorders, atherosclerosis, and cardiovascular disorders.

4. The method of claim **1**, wherein the mammal is a cholesterol-elevated mammal.

5. The method of claim **4**, wherein the test mammal has a disruption in both CAR alleles.

6. The method of claim **1,** wherein said cholesterol indicator is the level of serum cholesterol.

7. The method of claim **1**, wherein said cholesterol indicator is the level of a member selected from the group consisting of HDL cholesterol, LDL cholesterol, and VLDL cholesterol.

8. The method of claim **1**, wherein said cholesterol indicator is the mRNA level of a gene involved in the regulation of cholesterol levels.

9. The method of claim **1**, wherein said CAR-mediated intermolecular interaction is CAR-mediated gene expression.

10. The method of claim **9**, wherein the ability of said candidate therapeutic agent to modulate CAR-mediated gene expression is assessed by:

providing a cell that comprises:

a) a polynucleotide encoding a fusion polypeptide that comprises: 1) an amino acid sequence that comprises a DNA binding domain of a polypeptide; and 2) a ligand binding domain that is substantially identical to a ligand binding domain of CAR; and

b) a reporter gene construct which comprises a response element to which the DNA binding domain can bind, wherein the response element is operably linked to a promoter that is operative in the cell and the promoter is operably linked to a reporter gene; and

contacting said cell with said candidate therapeutic agent; and

determining whether said reporter gene is expressed at a higher or lower level in the presence of said candidate therapeutic agent as compared to expression in the absence of said candidate therapeutic agent.

11. The method of claim **10**, wherein said candidate therapeutic agent is 5β-pregnan-3,20-dione.

12. The method of claim **10**, wherein said DNA binding domain is substantially identical to a DNA binding domain from a polypeptide selected from the group consisting of: CAR, a GAL4 transcription factor, an estrogen receptor, a progesterone receptor, a glucocorticoid receptor, an androgen receptor, a mineralcorticoid receptor, a vitamin D receptor, a retinoid receptor, and a thyroid hormone receptor.

13. The method of claim **12,** wherein said DNA binding domain is a CAR DNA binding domain and the response element is a CAR response element.

14. The method of claim **13**, wherein said CAR response element is a DR-5 element or a DR-4 element.

15. The method of claim **10**, wherein said reporter gene encodes a marker protein selected from the group consisting of: luciferase, alkaline phosphatase, beta-galactosidase, chloramphenicol acetyltransferase and green fluorescent protein.

16. The method of claim **1**, wherein said CAR-mediated intermolecular interaction is the binding of a polypeptide that comprises a CAR ligand binding domain to a ligand for CAR.

17. The method of claim **16**, wherein said polypeptide is a CARα or a CARβ.

18. The method of claim **16**, wherein said ligand for CAR comprises a sensor peptide.

19. The method of claim **18**, wherein said ligand for CAR comprises a receptor binding domain of a coactivator or a corepressor.

20. The method of claim **19**, wherein said coactivator is SRC-1.

21. The method of claim **20**, wherein said sensor peptide is rhodamine labeled ILRKLLQE.

22. The method of claim **16**, wherein the binding of the polypeptide that comprises a CAR ligand binding domain to the ligand for CAR is determined in the presence of a naturally occurring ligand for CAR.

23. The method of claim **22**, wherein said naturally occurring ligand for CAR is 5β-pregnan-3,20-dione.

24. The method of claim **16**, wherein said method comprises determining whether said compound can inhibit the interaction between the CAR ligand binding domain and the CAR ligand.

25. The method of claim **24**, wherein said CAR ligand is labeled.

26. The method of claim **25**, wherein said CAR ligand is radiolabeled.

27. The method of claim **24**, wherein said CAR ligand is labeled with a fluorophore.

28. The method of claim **27**, wherein said method comprises a fluorescence polarization assay.

29. The method of claim **27**, wherein said method comprises a fluorescence resonance energy transfer assay.

30. The method of claim **27**, wherein said CAR is labeled with a fluorophore.

31. The method of claim **30**, wherein said method comprises a fluorescence resonance energy transfer assay or a fluorescence polarization assay.

32. The method of claim **24**, wherein said CAR ligand is selected from the group consisting of:

5α-androst-16-en-3α-ol, 5α-androst-16-en-3α-ol acetate, 5α-androstane-3α-ol, 5α-androst-16-en-3α-ol acetate and 5β-pregnan-3,20-dione.

33. A method for identifying a therapeutic agent for use in treating a CAR-mediated disorder or condition the method comprising:

administering a compound to a CAR compromised mammal; and

determining whether administration of the compound results in a change in cholesterol level compared to a mammal to which the compound is not administered.

34. The method of claim **33**, wherein the method further comprises administering the compound to a CAR non-compromised mammal and comparing the effect on the cholesterol level indicator of administering the compound to that of administering the compound to the CAR compromised mammal.

35. The method of claim **33**, wherein said cholesterol level indicator is the level of serum cholesterol.

36. The method of claim **33**, wherein said cholesterol level indicator is the level of a member selected from the group consisting of HDL cholesterol, LDL cholesterol, and VLDL cholesterol.

37. The method of claim **33**, wherein said cholesterol level indicator is the mRNA level of a gene involved in the regulation of cholesterol levels.

38. The method of claim **33**, wherein said CAR compromised mammal is a mammal having a disruption in both CAR alleles.

39. The method of claim **38**, wherein said CAR compromised mammal is a mouse.

40. The method of claim **38**, wherein said disruption occurs in the coding region for the DNA binding domain of CAR.

41. The method of claim **38**, wherein said disruption in a CAR allele comprises an insertion at codons for amino acid positions from about amino acid 21 to about amino acid 86 of CARβ.

42. A method for treating a CAR-mediated disorder or condition, the method comprising:

administering to a mammal having a CAR-mediated disorder or condition an effective amount of a therapeutic agent that modulates CAR-mediated regulation of cholesterol levels.

43. The method of claim **42**, wherein said therapeutic agent is identified by:

screening one or more compounds to determine whether said compounds can modulate a CAR-mediated intermolecular interaction;

administering the candidate therapeutic agent to a test mammal; and

determining whether the level of a cholesterol indicator is affected in said test mammal.

44. The method of claim **42**, wherein said CAR-mediated disorder or condition is selected from the group consisting of: hypercholesterolemia, lipid disorders, atherosclerosis, and cardiovascular disorders.

45. A non-human mammal having a genome that comprises a disruption in at least one CAR allele.

46. The non-human mammal of claim **45**, wherein said disruption comprises an insertion, deletion or mutation in a region of the CAR allele that encodes for a DNA binding domain of CAR.

47. The non-human mammal of claim **46**, wherein said disruption comprises an insertion at codons for amino acid positions from 21 to about 86 of CARβ.

48. A non-human mammal having a genome that comprises a disruption in both CAR alleles.

49. The non-human mammal of claim **48**, wherein said disruption comprises an insertion, deletion or mutation in a region of the CAR allele that encodes for a DNA binding domain of CAR.

50. The non-human mammal of claim **48**, wherein said disruption comprises an insertion at codons for amino acid positions from 21 to about 86 of CARβ.

51. The non-human mammal of claim **48**, wherein said non-human mammal exhibits an increased level of serum cholesterol relative to a wild-type mammal.

52. A method for producing a transgenic non-human mammal having a genome that comprises a disrupted CAR allele, the method comprising:

introducing into embryonic stem cells a polynucleotide that comprises a coding region for a portion of a CAR polypeptide, wherein the polynucleotide sequence includes a disruption in the coding region of a portion of said CAR polypeptide;

identifying a cell into which said polynucleotide sequence has been integrated into an endogenous CAR allele;

introducing said cell into a blastocyst, thereby forming a transgenic blastocyst;

implanting said transgenic blastocyst into a pseudopregnant mammal and allowing said pseudopregnant mammal give birth to a transgenic mammal.

53. The method of claim **52**, wherein said transgenic mammal contains a disrupted CAR allele in its germline.

54. The method of claim **53**, further comprising breeding said transgenic mammal to generate a heterozygous mammal comprising a disrupted CAR allele.

55. The method of claim **53**, further comprising mating a male and a female mammal each heterozygous for said disrupted CAR allele, to form progeny that are homozygous for said disrupted CAR allele.

56. The method of claim **52**, wherein said disrupted CAR allele comprises an insertion into a region of the CAR allele that codes for a DNA binding domain of CAR.

57. The method of claim **52**, wherein said disrupted CAR allele comprises an insertion at codons for amino acid positions from about 21 to about 86 of CARβ.

58. The method of claim **56**, wherein said insertion comprises a selectable marker gene.

59. The method of claim **58**, wherein said marker gene encodes for neomycin resistance.